

Characterization of *Aerococcus viridans* Isolates from Swine Clinical Specimens[▽]

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Received 22 January 2007/Returned for modification 11 April 2007/Accepted 19 June 2007

We present here the biochemical and genetic characterization and antimicrobial susceptibility of 58 isolates of *Aerococcus viridans* isolated in pure culture from different clinical specimens of normally sterile body sites of pigs. *A. viridans* isolates were commonly susceptible to β -lactam antimicrobials and exhibited a great genetic heterogeneity as determined by pulsed-field gel electrophoresis typing. The results indicate that *A. viridans* might be included in the list of possible etiological agents causing disease in pigs.

The genus *Aerococcus* was initially described including a single species, *Aerococcus viridans*. Five new species of *Aerococcus* have been further identified: *Aerococcus urinae*, *Aerococcus sanguinicola*, *Aerococcus christensenii*, *Aerococcus urinaequi*, and *Aerococcus urinaehominis* (6; <http://www.bacterio.net>). The clinical significance of these species has been clearly established for *A. urinae* and *A. viridans*. *A. urinae* is a rarely reported human pathogen that has been identified as responsible for urinary tract infections, septicemia, sepsis, endocarditis, or lymphadenitis (22–24, 32). *A. viridans* has been associated with different human infections such as endocarditis, urinary tract infections, arthritis, or meningitis (7, 10, 13, 20). Moreover, this species is a pathogen for crustaceans, causing gaffkemia in marine lobsters (1, 26), and it has been associated with septicemia in sea turtles (27). *A. viridans* has also been isolated from the milk of cows with subclinical mastitis (5) and has been associated with septicemia in immunodeficient mice (3); there have been no reports, as far as we know, of the isolation of this microorganism from other animal species. In the present study we report the isolation, biochemical and genetic characterization, and antimicrobial susceptibility of 58 isolates of *A. viridans* from different clinical specimens of pigs.

According to the source of isolation, 30 isolates were obtained from the joints of pigs with arthritis, 14 isolates were obtained from the brains of pigs with meningitis, and 14 isolates were obtained from the lungs of pigs with pneumonia. All isolates were recovered in pure culture from individual animal samples in moderate to high numbers, which is indicative of their clinical significance and therefore likely to be associated with bacterial infection. Details of the clinical background of the *A. viridans* isolates included in the study are given in Table 1. Isolation of *A. viridans* isolates from clinical specimens was carried out on Columbia agar containing 5% of defibrinated sheep blood (bioMérieux España, s.a.) after incubation at 37°C for 24 h under aerobic and anaerobic conditions. All isolates were gram-positive catalase-negative cocci arranged in single

cells, in pairs, in tetrads, or in small groups. The organisms were facultatively anaerobic and produced unpigmented, alpha-hemolytic, circular, colonies <1 mm in diameter after 24 h of incubation at 37°C on blood agar. Biochemical identification of *A. viridans* isolates was carried out by using the commercial identification system API Rapid ID32 Strept (bioMérieux España, s.a.) according to the manufacturer's instructions. Identification was achieved after 24 h by using the corresponding

TABLE 1. Details of the 58 strains of *A. viridans* examined

Herd	Strain(s)/yr isolation-clinical process ^a	PFGE profile ^b
1	65/99-M; 132/99-M; 140/99-M	14; 17; 12
2	169/99-M; 246/00-M	49; 10
3	506/02-M; 511/02-M; 905/02-P; 1318/02-P	27; 41; 29; 48
4	994/02-M	23
5	11/03-M	21
6	162/02-P; 206/02-M	6; 8
7	C 769/03-A; C 771/03-M	31; 11
8	424/01-M; 1792/02-P; 1977/02-A; 2185/02-A; 2303/02-A; 420/03-A; 421/03-A; C 1537/03-M	39; 2; 20; 1; 50; 52; 32; 45
9	C 916/04-M; C 917/04-P	28; NT ^c
10	C 913/04-M	30
11	2068/03-P	NT
12	594/01-P; 595/01-A; 607/01-A	46; NT; 43
13	2162/02-P	26
14	630/01-P; 1544/02-A; 1545/02-A; 1546/02-A; 1547/02-A; 1549/02-A; C 637/03-A; C 638/03-A; C 642/03-A;	22; 5; 34 (2); 9; 49; 15; 44; 18
15	C 575/04-P	NT
16	1286/02-P	4
17	427/01-P	42
18	C 345/04-P; C 346/04-P	13 (2)
19	552/01-A	3
20	609/01-A	51
21	539/01-A; 540/01-A; 541/01-A	53; 7; 35
22	250/03-A; C 1172/03-A; C 1437/03-A	40; 19; 24
23	C 1950/03-A	38
24	C 215/04-A	33
25	C 856/04-A	25
26	C 849/04-A; C 850/04-A	37; 16

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[▽] Published ahead of print on 27 June 2007.

^a A, P, and M, clinical isolates were from animals with arthritis, pneumonia, and meningitis, respectively.

^b Data in parentheses indicate the number of strains with the same PFGE profile.

^c NT, nontypeable.

TABLE 2. Numerical codes and discrepant biochemical tests for the 14 *A. viridans* isolates that were not accurately identified with the API Rapid ID32 Strept system

Isolate	Numerical code	Discrepant biochemical tests ^a
C 771/03	22022111110	Man; Lac; Appa
C 913/04	23000111010	Man; Lac; Tre
C 637/03	63630011130	Sor; Tag; β -Gar; Pyra
630/01	42050011100	Man; Tre; β -Gar; Pyra
2162/02	23371115110	VP; Mlz
540/01	02220011100	Lac; Pyra
607/01	23000011100	Man; Lac; Tre
421/03	60010011100	Man; Tre; β -Gar; Pyra
C 849/04	20010011110	Man; Tre; Pyra
C 850/04	21220011100	Lac; Pyra
905/02	20010011100	Man; Tre; Pyra
506/02	00210011110	Tre; Pyra
1547/02	63260015110	Lac; Mlz; β -Gar; Pyra
C 575/04	63370111170	Tag; β -Gar; β -Man

^a Man, Mlz, Sac, Tre, Lac, Sor, and Tag correspond to the acidification of mannitol, melzitose, sucrose, trehalose, lactose, sorbitol, and tagatose, respectively; Appa, β -Gar, β -Man, and Pyra, correspond to the production of alanyl-phenylalanyl-proline- β -arylamidase, β -galactosidase, β -mannosidase, and pyroglutamic acid arylamidase enzymes, respectively; VP, acetoin production (Voges-Proskauer test).

identification software (version 2.0). A total of 75.9% ($n = 44$) of the isolates examined in the present study were accurately identified (excellent or very good identification) by using this identification system, although a considerable phenotypic diversity was observed with 36 different biochemical profiles. In addition, two isolates (3.4%) yielded an unacceptable biochemical profile for *A. viridans*, five isolates (8.6%) were doubtfully identified as *A. viridans*, and seven isolates (12.1%) were identified with a low discrimination level between *A. viridans* and *Streptococcus acidominimus*. The failure of the API Rapid ID32 system to correctly identify *A. viridans* was not unexpected because it has been shown that commercial available phenotypic identification systems are not completely reliable for the accurate identification of aerobic gram-positive catalase-negative cocci (2). The phenotypic misidentification of *A. viridans* with *S. acidominimus* agrees also with previous reports (8). Most of the isolates that were not correctly identified as *A. viridans* with the API Rapid ID32 system failed to produce pyroglutamic acid arylamidase and failed to produce acid from mannitol, lactose, and trehalose. Details of the nu-

merical profiles and discrepant tests obtained with these *A. viridans* isolates are given in Table 2.

The identification of the 58 isolates was later confirmed by PCR and sequencing of their 16S rRNA gene. A 540-bp 16S rRNA gene fragment was amplified by PCR using the *A. viridans* species-specific primers AC2 (5'-GTGCTTGCACTTCTGACGTTAGC-3') and AC4 (5'-TGAGCCGTGGGCTTTTCACAT-3') (12). All of the isolates gave the expected amplification product of 540 bp, which is specific for *A. viridans*. For sequencing of the 16S rRNA gene, each isolate was amplified by PCR and further sequenced as described previously (29). The determined 16S rRNA sequences consisted of about 1,400 nucleotides and were compared to the sequences of other gram-positive, catalase-negative species available in the GenBank and EMBL databases by using the FASTA program. Sequencing results resulted in 99.4 to 100% 16S rRNA gene sequence similarity with *A. viridans* ATCC 11536^T (accession no. M58797).

Limited data are available on the antimicrobial susceptibility of *A. viridans* clinical isolates. Thus, the antimicrobial susceptibility to different commonly used antimicrobials was determined by the disk diffusion method using commercially (Oxoid, Ltd.) prepared antimicrobial disks (Table 3). Inocula were prepared from a 48-h Columbia blood agar plate by suspending four colonies in 5 ml of Mueller-Hinton broth and adjusted to a 0.5 McFarland standard. The disk diffusion test was performed as described by the Clinical and Laboratory Standards Institute (CLSI; formerly the National Committee for Clinical Laboratory Standards) (16) using Mueller-Hinton plates (Oxoid) supplemented with 5% defibrinated sheep blood. The agar plates were examined after 24 h of incubation at 37°C. *Staphylococcus aureus* ATCC 25923 and *Escherichia coli* ATCC 25922 were included as quality controls. Since no specific inhibition zone diameter (IZD) breakpoints for aerococci are available, the IZD breakpoints for resistance to clindamycin, tetracycline, ceftiofur, and gentamicin were those recommended by the CLSI (17) for testing veterinary gram-positive microorganisms. For penicillin, vancomycin, ampicillin, and erythromycin, the IZD breakpoints used were those recommended for testing animal enterococci, and the breakpoints for amoxicillin-clavulanic acid and chloramphenicol were those recommended for testing animal staphylococci and streptococci, respectively (17). For ciprofloxacin the IZD breakpoint used was that recommended previously for testing *Staphylo-*

TABLE 3. In vitro susceptibility as determined by the disk diffusion method for the 58 *A. viridans* isolates

Antimicrobials (amt)	IZD breakpoints	No. of isolates with an IZD (mm) of ^a :															Resistant strains (%)
		7-8	9-10	11-12	13-14	15-16	17-18	19-20	21-22	23-24	25-26	27-28	29-30	31-32	33-34	35-36	
Penicillin (10 U)	≤ 14							3	6	9	14	6	10	3	3	3	0
Ampicillin (10 μ g)	≤ 16										4	6	12	14	3	10	0
Amoxicillin-clavulanic acid (20/10 μ g)	≤ 19										2	1	4	9	9	11	0
Ceftiofur (30 μ g)	≤ 17						2	1	4	6	5	3	11	9	8	7	0
Streptomycin (10 μ g)	≤ 11	48	3	5	2												89.6
Gentamicin (10 μ g)	≤ 12	22			4	11	11	4	2	3	1						37.9
Erythromycin (15 μ g)	≤ 13	32	2							2	2	5	6	8	1		58.6
Clindamycin (2 μ g)	≤ 14	39		6	1	1	1		3		3	2		1			79.3
Vancomycin (30 μ g)	≤ 14							26	21	8	2		1				0
Ciprofloxacin (5 μ g)	≤ 15	11			1	7	10	21	6	2							20.7
Chloramphenicol (30 μ g)	≤ 17		2	6	8	4		6	13	11	5	3					34.5
Tetracycline (30 μ g)	≤ 12	53	2		2						1						94.8

^a In order to present clearer data, the IZDs have been grouped into 2-mm intervals.

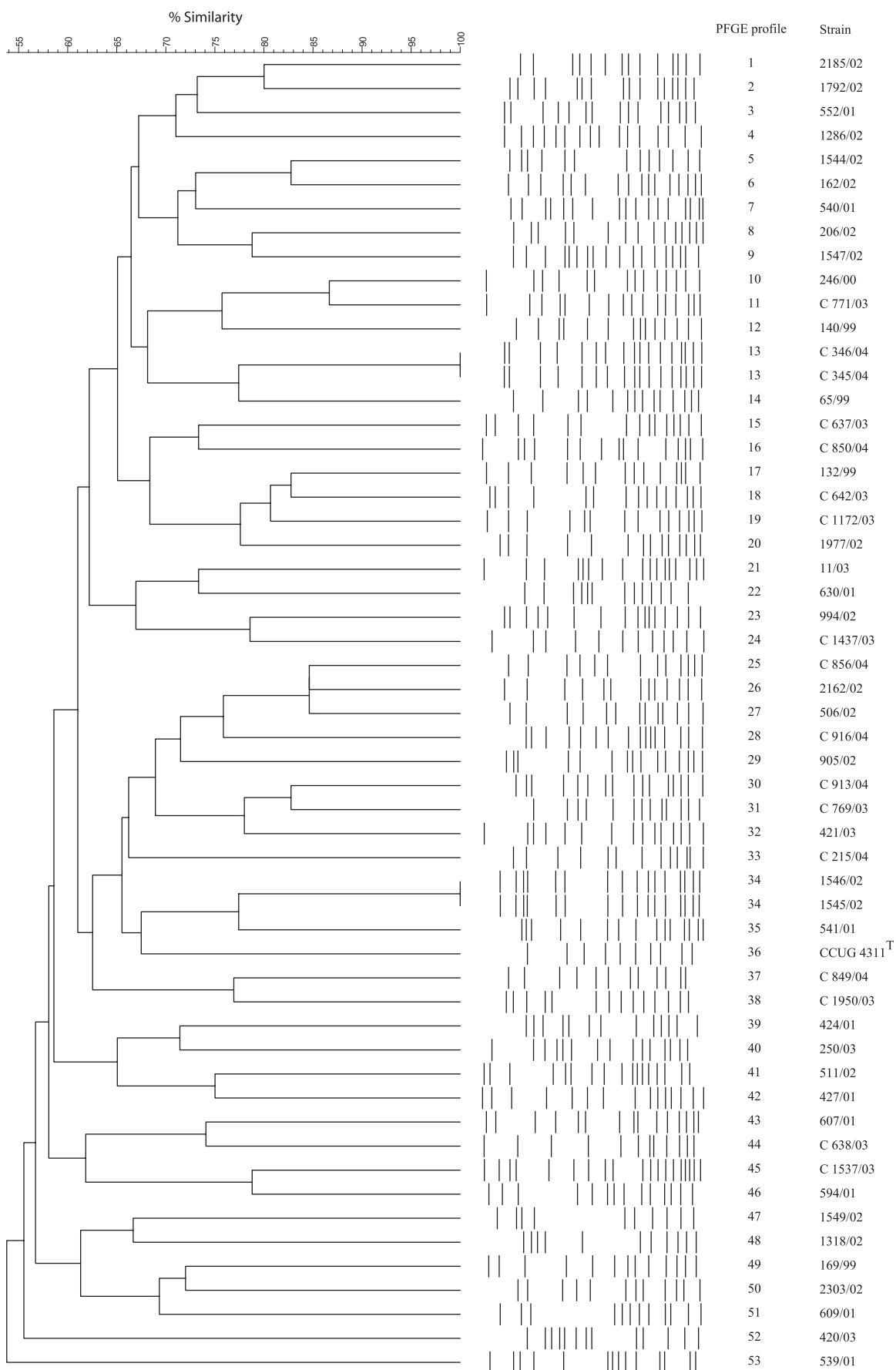


FIG. 1. Dendrogram showing the genetic relationship, based on UPGMA cluster analysis, of *A. viridans* isolates examined in the present study.

coccus (15), and for streptomycin the IZD breakpoint used was the one recommended by the CLSI (14). The results of the susceptibility testing of the clinical isolates of *A. viridans* as distribution of the IZDs values and percentage of resistant isolates are shown in Table 3. Although β -lactam-resistant *A. viridans* strains can be isolated from human clinical specimens (31), all isolates included in the present study were susceptible to β -lactam antimicrobials (penicillin, ampicillin, amoxicillin-clavulanic acid, and ceftiofur), as well as to vancomycin. These results are in agreement with the susceptibility pattern observed for cow isolates (19). On the other hand, a high frequency of resistance was found to tetracycline, clindamycin, and streptomycin, with more than 79% of the *A. viridans* isolates being resistant to these antimicrobials. The resistance of *A. viridans* to the other antimicrobials tested was variable, ranging from 20.7% (ciprofloxacin) to 58.6% (erythromycin). Resistance to ampicillin, chloramphenicol, gentamicin, or fluoroquinolones has also been reported occasionally for human clinical strains (31).

Isolates of *A. viridans*, as well as of the type strain of this bacterial species (*A. viridans* CCUG 4311^T), were genetically characterized by pulsed-field gel electrophoresis (PFGE) with the restriction enzyme *Sma*I (MBI Fermentans, Vilnius, Lithuania) as described previously (28). The PFGE patterns were examined visually, and similarities between restriction endonuclease digestion profiles were analyzed by using BioNumerics software (Applied Maths, Belgium). A similarity matrix was computed and transformed into an agglomerative cluster using the unweighted pair group method with arithmetic averages (UPGMA) (25). All but four isolates (93.2%) were successfully characterized by PFGE after genomic DNA digestion; 53 PFGE profiles were identified. The genetic relationship between the *A. viridans* isolates on the basis of the *Sma*I-digested DNA restriction patterns after the UPGMA clustering is shown in Fig. 1. Overall, *A. viridans* exhibited a great genetic heterogeneity, with the 92.7% of PFGE profiles being represented by single isolates. Only two isolates (isolates 1545/02 and 1546/02 from farm 14 and isolates C 345/04 and C 346/04 from farm 18; Table 1) isolated in the same year from two different pigs at the two farms exhibited undistinguishable PFGE profiles. In most cases, the number of *A. viridans* isolates per herd ranged between 1 and 3 (Table 1), but in three herds in which more than four isolates were isolated (nine, eight, and four isolates in herds 14, 8, and 3, respectively) at least three different PFGE profiles were identified. *A. viridans* is a usual commensal microorganism of the normal flora, and it is also widespread in the environment (4, 30), which could explain the great number of different PFGE profiles obtained among the isolates studied.

All of the *A. viridans* isolates were isolated from normally sterile body sites. Most of them were isolated from joints (51.7%) with clinical signs of arthritis, followed by brains and lungs (24.1% each) with clinical signs of meningitis and pneumonia, respectively. Our overall frequencies of isolation of *A. viridans* from pigs with arthritis, pneumonia, and meningitis were about 11.5, 1, and 2.2% of the isolates, respectively (A. I. Vela, unpublished data). Arthritis, meningitis, and pneumonia in pigs can be associated with infections of different microorganisms, such as *Streptococcus suis*, *Haemophilus parasuis*, *Actinobacillus pleuropneumoniae*, *Pasteurella multocida*, *Mycoplasma hyopneumoniae*, *Mycoplasma hyosynoviae*, or *E. coli*

(21), but none of these bacterial pathogens was isolated in our laboratory during the routine microbiology processing of the clinical samples from which *A. viridans* was recovered. These data and the fact that all of the *A. viridans* isolates were recovered in pure culture greatly support a causal relationship between this microorganism and lesions. However, the great genetic diversity of this microorganism observed in the present study, as well as the isolation of clinical isolates with different PFGE profiles from different pigs of the same herd, indicate an exposure of animals to multiple *A. viridans* strains, which is in accordance with its wide distribution (4, 30), and also suggests an opportunistic pathogenic character of this microorganism. The pathogenicity of *A. viridans* is not clearly understood and immunosuppression seems to contribute or to be necessary for the development of clinical disease (3). Also, it is well known that in most infectious diseases of swine, environmental and management factors play a crucial role in the clinical expression of disease (11). All of the herds from which *A. viridans* was isolated were managed under "intensive" conditions, and most of them also had previous records of infection by porcine respiratory and reproductive syndrome virus (PRRSV). It has been suggested that significant PRRSV-induced immunosuppression might well occur under practical farm conditions, predisposing the animals to bacterial infections (9, 18). Therefore, it is possible that a previous long-term exposure to PRRSV and/or other predisposing environmental farm conditions, such as overcrowding or poor ventilation, that can cause stress and subsequent immune suppression may have favored the infection with this opportunistic pathogen.

To the best of our knowledge, this is the largest study of isolates of *A. viridans* from clinical specimens in animals. It would appear from the results of the study that *A. viridans* is an opportunistic pathogen organism that should be included in the list of possible etiological agents causing disease in pigs, principally as responsible for arthritis.

A.I.V. has a fellowship from the Ramon y Cajal Program (Spanish Ministry of Science and Technology/U.C.M.). J.C. was a recipient of a predoctoral grant from the Spanish Ministry of Education. This study was partially funded by project AGL2003-08848-C02-01/GAN of the Spanish Ministry of Science and Technology.

REFERENCES

1. Battison, A. L., R. J. Cawthorn, and B. Horney. 2004. Response of American lobsters *Homarus americanus* to infection with a field isolate of *Aerococcus viridans* var. *homari* (*Gaffkemia*): survival and hematology. *Dis. Aquat. Organ.* **61**:263–268.
2. Bosshard, P. P., S. Abels, M. Alwegg, E. C. Böttger, and R. Zbinden. 2004. Comparison of conventional and molecular methods for identification of aerobic catalase-negative gram-positive cocci in the clinical laboratory. *J. Clin. Microbiol.* **42**:2065–2073.
3. Dagnaes-Hansen, F., M. Kilian, and K. Fursted. 2004. Septicaemia associated with an *Aerococcus viridans* infection in immunodeficient mice. *Lab. Anim.* **38**:321–325.
4. Deibel, R. H., and C. F. Niven. 1960. Comparative study of *Gaffkya homari*, *Aerococcus viridans*, tetrad-forming cocci from meat curing brines, and the genus *Pediococcus*. *J. Bacteriol.* **79**:175–180.
5. Devriese, L. A., J. Hommez, H. Laevens, B. Pot, P. Vandamme, and F. Haesebrouck. 1999. Identification of aesculin-hydrolyzing streptococci, lactococci, aerococci, and enterococci from subclinical intramammary infections in dairy cows. *Vet. Microbiol.* **70**:87–94.
6. Euzéby, J. P. 1997. List of bacterial names with standing in nomenclature: a folder available on the Internet. *Int. J. Syst. Bacteriol.* **47**:590–592.
7. Facklam, R. R., and J. A. Elliot. 1995. Identification, classification, and clinical relevance of catalase-negative, gram-positive cocci, excluding the streptococci and enterococci. *Clin. Microbiol. Rev.* **8**:470–495.
8. Facklam, R., M. Lovgren, P. L. Shewmaker, and G. Tyrrell. 2003. Phenotypic

- description and antimicrobial susceptibilities of *Aerococcus sanguinicola* isolates from human clinical samples. *J. Clin. Microbiol.* **41**:2587–2592.
9. Galina, L., C. Pijoan, M. Sitjar, W. T. Christianson, K. Rossow, and J. E. Collins. 1994. Interaction between *Streptococcus suis* serotype 2 and porcine reproductive and respiratory syndrome virus in specific pathogen-free piglets. *Vet. Rec.* **134**:60–64.
 10. Gopalachar, A., R. L. Akins, W. R. Davis, and A. A. Siddiqui. 2004. Urinary tract infection caused by *Aerococcus viridans*, a case report. *Med. Sci. Monit.* **10**:CS73–CS75.
 11. Gradner, I. A., and P. C. Blanchard. 2006. Interpretation of laboratory results, p. 219–239. In B. E. Straw, J. J. Zimmerman, S. D'Allaire, and D. J. Taylor (ed.), *Diseases of swine*, 9th ed. Blackwell Publishing, New York, NY.
 12. Grant, K. A., J. H. Dickinson, M. D. Collins, and R. G. Kroll. 1992. Rapid identification of *Aerococcus viridans* using the polymerase chain reaction and an oligonucleotide probe. *FEMS Microbiol. Lett.* **74**:63–69.
 13. Nathavitharana, K. A., S. N. Arseculeratne, H. A. Aponso, R. Vijeratnam, L. Jayasena, and C. Navaratnam. 1983. Acute meningitis in early childhood caused by *Aerococcus viridans*. *BMJ (Clin. Res. Ed.)*, **286**:1248.
 14. National Committee for Clinical Laboratory Standards. 1994. Performance standard for antimicrobial susceptibility testing; 5th informational supplement. NCCLS document M100–S5. NCCLS, Villanova, PA.
 15. National Committee for Clinical Laboratory Standards. 2002. Performance standards for antimicrobial susceptibility testing; 12th informational supplement. NCCLS document M100–S12. NCCLS, Wayne, PA.
 16. National Committee for Clinical Laboratory Standards. 2002. Performance standards for antimicrobial disk and dilution susceptibility test for bacteria isolated from animals: approved standard, 2nd ed. NCCLS document M31–A2. NCCLS, Wayne, PA.
 17. National Committee for Clinical Laboratory Standards. 2004. Performance standards for antimicrobial disk and dilution susceptibility test for bacteria isolated from animals: informational supplement. NCCLS document M31–S1. NCCLS, Wayne, PA.
 18. Oleksiewicz, M. B., and J. Nielsen. 1999. Effect of porcine reproductive and respiratory syndrome virus (PRRSV) on alveolar lung macrophage survival and function. *Vet. Microbiol.* **66**:15–27.
 19. Owens, W. E., J. L. Watts, B. B. Greene, and C. H. Ray. 1990. Minimum inhibitory concentrations and disk diffusion zone diameter for selected antibiotics against streptococci isolated from bovine intramammary infections. *J. Dairy Sci.* **73**:1225–1231.
 20. Popescu, G. A., E. Benea, E. Mitache, C. Piper, and D. Horstkotte. 2005. An unusual bacterium, *Aerococcus viridans*, and four cases of infective endocarditis. *J. Heart Valve Dis.* **14**:317–319.
 21. Quinn, P. J., M. E. Carter, B. Markey, and G. R. Carter. 1999. A system approach to infectious diseases on a species basis, p. 497–614. In P. J. Quinn, M. E. Carter, B. Markey, and G. R. Carter (ed.), *Clinical veterinary microbiology*. Mosby, Edinburgh, United Kingdom.
 22. Santos, R., E. Santos, S. Goncalves, A. Marques, J. Sequeira, P. Abecasis, and M. Cadete. 2003. Lymphadenitis caused by *Aerococcus urinae* infection. *Scand. J. Infect. Dis.* **35**:353–354.
 23. Schuur, P. M., L. Sabbe, A. J. van der Wou, G. J. Montagne, and A. G. Buiting. 1999. Three cases of serious infection caused by *Aerococcus urinae*. *Eur. J. Clin. Microbiol. Infect. Dis.* **18**:368–371.
 24. Sierra-Hoffman, M., K. Watkins, C. Jinadatha, R. Fader, and J. L. Carpenter. 2005. Clinical significance of *Aerococcus urinae*: a retrospective review. *Diagn. Microbiol. Infect. Dis.* **53**:289–292.
 25. Sneath, P. H., and R. R. Sokal. 1973. Numerical taxonomy. W. H. Freeman & Co., San Francisco, CA.
 26. Stewart, J. E., J. W. Cornick, B. M. Zwicker, and B. Arie. 2004. Studies on the virulence of *Aerococcus viridans* (var.) *homari*, the causative agent of gaffkemia, a fatal disease of homarid lobsters. *Dis. Aquat. Organ.* **60**:149–155.
 27. Torrent, A., S. Deniz, A. Ruiz, P. Calabuig, J. Sicilia, and J. Oros. 2002. Esophageal diverticulum associated with *Aerococcus viridans* infection in a loggerhead sea turtle (*Caretta caretta*). *J. Wildl. Dis.* **38**:221–223.
 28. Vela, A. I., J. Goyache, C. Tarradas, I. Luque, A. Mateos, M. A. Moreno, C. Borge, J. A. Perea, L. Dominguez, and J. F. Fernández-Garayzábal. 2003. Analysis of genetic diversity of *Streptococcus suis* clinical isolates from pigs in Spain by pulsed-field gel electrophoresis. *J. Clin. Microbiol.* **41**:2498–2502.
 29. Vela, A. I., N. García, M. V. Latre, A. Casamayor, C. Sánchez-Porro, V. Briones, A. Ventosa, L. Domínguez, and J. F. Fernández-Garayzábal. *Aerococcus suis* sp. nov., isolated from clinical specimens from swine. *Int. J. Syst. Evol. Bacteriol.* **57**:1291–1294.
 30. Woodward, W. D., A. C. Ward, L. K. Fox, and L. B. Corbeil. 1988. Teat skin normal flora and colonization with mastitis pathogen inhibitors. *Vet. Microbiol.* **17**:357–365.
 31. Young, Uh., J. Seog Son, I. H. Jang, K. J. Yoon, and S. K. Hong. 2002. Penicillin-resistant *Aerococcus viridans* bacteremia associated with granulocytopenia. *J. Korean Sci.* **17**:113–115.
 32. Zhang, Q., C. Kwok, S. Attorri, and J. E. Clarridge. 2000. *Aerococcus urinae* in urinary tract infections. *J. Clin. Microbiol.* **38**:1703–1705.